SUDDEN APPARENTLY UNEXPLAINED DEATH DURING INFANCY

I. Pathologic Findings in Infants Found Dead *

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The purpose of this paper is to describe the morphologic findings in infants found dead while in apparent good health, in whom the gross necropsy findings were insufficient to explain death.

This report will be illustrated largely by our observations on 31 unselected consecutive cases studied during 1950 (Table I), occurring in the Borough of Queens, New York City (population 1,500,000). The material to be described is part of a series of 299 cases of infants less than 1 year of age, found dead while in apparent good health, investigated by us during the past 20 years (1931-1951). The 299 cases may be grouped into two categories: a smaller series, comprising 16.8 per cent of the total, with lesions that are conventionally regarded as establishing a cause of death, such as congenital heart disease, meningitis, (grossly) purulent laryngotracheobronchitis, and pneumonia; and a larger group, representing 83.2 per cent of the total, consisting of infants whose deaths remained unexplained at the conclusion of the necropsy.

This larger group constitutes an important medicolegal as well as pediatric problem. These cases are often certified as dying of accidental mechanical suffocation, aspiration of vomitus, and status thymicolymphaticus, when microscopic studies have not been made or have been erroneously interpreted. The unsatisfactory nature of the diagnosis of status thymicolymphaticus has long been established.[1] Aspiration of vomitus is not an uncommon finding in subjects dying of other causes.[2] It should, therefore, be regarded as an agonal phenomenon, with rare exceptions. We and others[3-5] have shown that the allegation of accidental mechanical suffocation by posture or ordinary bedclothes is unsupported by any real evidence. Critical examination of the circumstances surrounding the discovery of the death shows that in only a small proportion of the cases (20 per cent) was the infant allegedly found in the face-down position; or covered by bed-

* The second and third papers of the series entitled "Pathologic Findings in Infants Observed to Die Suddenly" and "Pathologic Findings in Infants Dying Immediately after Violence, Contrasted with Those in Sudden Apparently Unexplained Death," respectively, are to appear in the succeeding issue of this Journal.

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TABLE I

Summary of Findings in 31 Consecutive Infants Found Dead While in Apparent

Case	Age and sex	Position when found	Exposure to respiratory disease	Prior history	Gross findings
No. 1 50-A-6	6 wks. Male	On abdomen, face down in crib	Positive	Stuffy nose for several days	Purulent exudate in left mastoid; lungs: 105 gm., congestion and edema; bronchi congested, contained mucoid material; pharynx, epiglottis, and larynx fiery red
No. 2 50-A-51	3 mos. Male	On abdomen, face to side, in crib	Positive	"Sniffling" 1 week, more marked the night before death	Mucoid exudate in mastoids; lungs: 140 gm., congestion and edema; bronchi congested, contained mucoid material
No. 3 50-A-54	6 wks. Male	On abdomen, face down in crib	Positive; also had exposure to measles	"Cold" for 2 weeks	Purulent exudate in mastoids; lungs: 100 gm., congestion and edema; bronchi congested
No. 4 ME 50-11	7 wks. Female	On abdomen, face to side, in carriage; blood on sheet	Positive	Had a running nose	Serous exudate in mastoid; lungs: 120 gm., congestion and edema; bronchi congested
No. 5 ME 50-14	3 wks. Female	No information	Positive	"Cold" 1 day	Normal mastoid; lungs: congestion and edema; bronchi contained mucoid material, mucosa congested
No. 6 ME 50-28	9 mos. Male	At foot of carriage, blanket over head	Positive	"Cold" for several days; breathed heavily the night before	Mucoid exudate in mastoids; lungs: 170 gm., congestion and edema
No. 7 ME 50-30	2 mos. Male	In mother's bed, face to side	None	"Light head cold" for 4 days	Mucoid exudate in mastoids; lungs: 110 gm., congestion and edema; bronchi contained mucoid material, congested
No. 8 ME 50-47	3 wks. Female	In parent's bed, between them, on back	(Sibling developed a cold the day after death)	"Mild" cold a few days before	Normal mastoid; lungs: 85 gm., congestion and edema
No. 9 ME 50-67	6 wks. Male	In crib, on abdomen, face to side	"Boarder" in hospital	None	Normal mastoid; lungs: 100 gm., congestion and edema; trachea and bronchi congested
No. 10 ME 50-71	12 mos. Female	In crib, on back	No information	"Cold" for 2 days; extensive eczema	Purulent exudate in mastoids; lungs: 175 gm., congestion and edema; bronchi congested and contained mucoid material; eczematoid dermatitis. (Boric acid, 0.15 mg. in 12 gm. formalin-fixed liver)
No. 11 ME 50-74	6 wks. Male	In crib, on back, head turned to one side	Positive; chicken pox contact; twin developed a running nose	Stuffy nose the evening before	Mucoid exudate in right mastoid; lungs: 100 gm., congestion and edema

Good Health and in Whom Death Remained Unexplained at Necropsy

Microscopic findings			Bacteriologic findings
Upper respiratory tract	Lung	Other significant findings	
Suppurative mastoiditis; non-suppurative laryngotracheitis	Congestion, edema, hemorrhage and collapse; bronchitis	Thrombosis in meningeal vessel; focal adrenal hemorrhages; focal glomerulitis; edema and cellular infiltration of the testis	Mastoid, larynx and trachea: hemolytic *Staphylococcus aureus;* lung and spleen, sterile
Suppurative mastoiditis; laryngotracheitis with early suppuration	Congestion, edema, hemorrhage and collapse; bronchitis	Thrombosis in cerebral vessels	Lung and spleen, sterile
Suppurative mastoiditis and tonsillitis; non-suppurative tracheitis and laryngitis with early necrosis	Edema, congestion, collapse and hemorrhage; early bronchitis	Thrombosis in pharynx and pituitary body	Mastoids, bronchi and liver: hemolytic *Staph. aureus;* lung and spleen, sterile
Suppurative laryngitis and tonsillitis with abscess	Hemorrhage, edema, congestion and collapse; early bronchitis; single minute focus of bronchopneumonia	Thrombosis in tonsil; focal adrenal hemorrhage	No pathogens; lung and spleen, sterile
Suppurative mastoiditis and laryngitis	Focal bronchopneumonia; hemorrhage, collapse, congestion and edema; bronchitis with early suppuration	Thrombosis in brain, pharynx, larynx, heart, thymus, lung; focal adrenal hemorrhage; mononuclear infiltration of parotid gland	Mastoid and trachea; *Streptococcus haemolyticus*
Suppurative mastoiditis, laryngitis, and tonsillitis with abscess	Focal bronchopneumonia; congestion, edema, collapse and hemorrhage; bronchitis	Thrombi in mesentery; glomerulonephritis, marked; focal adrenal hemorrhage	
Suppurative inflammation of mastoids; suppurative tracheitis, tonsillitis, laryngitis with necrosis	Focal bronchopneumonia; congestion, edema, collapse and hemorrhage; bronchitis with early suppuration	Thrombosis in deep laryngeal vessel; prominent periportal lymphocytic infiltration in liver; focal adrenal hemorrhage	Bronchus: hemolytic *Staph. aureus*
Non-suppurative inflammation of mastoids, larynx, and trachea	Hemorrhage, congestion, edema and collapse; moderate number of intra-alveolar squames (vernix)	Thrombosis in larynx and lung; focal adrenal hemorrhage	No pathogens; lung, sterile
Non-suppurative mastoiditis and laryngitis; suppurative tonsillitis with necrosis	Congestion, edema, hemorrhage and collapse; bronchitis with early suppuration; focal bronchopneumonia	Focal adrenal hemorrhage	Lung: hemolytic *Staph. aureus*
Suppurative mastoiditis, laryngitis, tracheitis, and tonsillitis	Congestion, edema, collapse and hemorrhage; bronchitis with early suppuration; focal interstitial pneumonitis	Thrombosis in brain and lung	Numerous bacteria in section of lung but no pathogens on culture
Pharyngitis with early suppuration	Hemorrhage, congestion, edema and collapse; bronchitis; minute focal bronchopneumonia	Thrombosis in brain, pharynx, and lung; focal glomerulitis; focal adrenal hemorrhage; infiltration of submaxillary and testis	Bronchus: hemolytic *Staph. aureus*

TABLE I (Continued)

Case	Age and sex	Position when found	Exposure to respiratory disease	Prior history	Gross findings
No. 12 ME 50-77	10 days Male	In crib, on abdomen, head not covered	None	None	Normal mastoid; lungs: 75 gm., congestion and edema
No. 13 ME 50-99	1 mo. Female	In crib, on back, bloody fluid oozing from nose	A sibling developed a cold after the death	None	Normal mastoid; lungs: 105 gm., congestion and edema
No. 14 ME 50-120	2 mos. Female	On abdomen, in carriage, small "blood" stain on sheet	Negative	None	Normal mastoid; lungs: congestion and edema; bronchi congested; minimal aspiration of stomach contents
No. 15 ME 50-133	5 days Male	Face down, in crib	Positive	None	Normal mastoid; lungs: congestion and edema
No. 16 ME 50-135	3 mos. Male	Face down, in crib	Negative	Occasional twitching for 2 days	Purulent exudate in mastoids; lungs: congestion and edema; minimal aspiration of stomach contents
No. 17 ME 50-141	5 mos. Male	In carriage, on abdomen, face to side	Positive	Restless and had a stuffy nose the night before	Mucoid exudate in mastoids; lungs: congestion and edema
No. 18 ME 50-143	1 mo. Male	In crib, face down; spot of blood on mattress	Positive	Loose stool the night before	Serous exudate in right mastoid; lungs: congestion
No. 19 ME 50-159	3 mos. Female	On abdomen, face to side, in crib	Positive	None	Normal mastoid; lungs: 90 gm., emphysema; minimal aspiration of stomach contents
No. 20 ME 50-168	2 mos. Male	Face down, in crib	Positive	Nose "stuffed up" the night before	Purulent exudate in mastoids; lungs: congestion and edema
No. 21 ME 50-176	4½ mos. Female	Face down, on abdomen, in carriage	Negative	None	Normal mastoid; lungs: 130 gm., emphysema; bronchi contained mucoid material; minimal aspiration of stomach contents
No. 22 ME 50-192	3 mos. Male	Face down, in crib	Positive	Mild cold 3 weeks before	Normal mastoid; lungs: 125 gm., congestion and edema; smaller bronchi contained thick gray mucoid material
No. 23 ME 50-194	2 mos. Male	On abdomen, in carriage, face to side	Negative	Running nose for 1 day, 2 weeks before	Mucoid exudate in mastoids; lungs: 160 gm., congestion and edema; bronchi congested and contained mucoid material
No. 24 ME 50-212	1 mo. Male	On abdomen, in crib, face to side	Positive	Running nose for 1 day, 2 weeks before	Normal mastoids; lungs: 90 gm., congestion; smaller bronchi contain tenacious mucus

Microscopic findings			Bacteriologic findings
Upper respiratory tract	Lung	Other significant findings	
Normal mastoids; suppurative laryngitis, with necrosis	Focal bronchopneumonia; hemorrhage, congestion, edema and collapse; few squames	Focal adrenal thrombosis and hemorrhage; focal epicardial infiltration	Spleen: *Staph. aureus;* meninges: *Strep. haemolyticus;* umbilicus: bacillus Friedlander, type A
Purulent mastoiditis; non-suppurative tracheitis	Hemorrhage, congestion, edema and collapse; bronchitis	Glomerulitis; focal adrenal hemorrhage	Lung and spleen, sterile
Non-suppurative mastoiditis; laryngitis and tracheitis, with necrosis	Congestion, hemorrhage, edema and collapse; bronchitis	Thrombosis in brain and larynx; focal adrenal hemorrhage; perineural lymphocytic infiltration in hilar region of lung	Lung and spleen, sterile
Normal mastoids; laryngitis with necrosis; non-suppurative tracheitis	Focal bronchopneumonia; congestion, edema, hemorrhage and collapse; bronchitis; moderate number of squames (vernix)	Thrombosis in brain, thymus and lung; adrenal hemorrhage; submaxillary and testicular infiltration; focal myocardial infiltration; interstitial edema in pancreas	No pathogens
Laryngitis with necrosis; non-suppurative tracheitis	Edema, hemorrhage, congestion and collapse; bronchitis	Thrombosis in lungs; focal adrenal hemorrhage; coronary sclerosis; focal epicardial infiltration	Mastoids: *Strep. haemolyticus*
Suppurative laryngitis	Edema, hemorrhage, congestion and collapse; early suppurative bronchitis	Lymphocytic infiltration of testis	
Non-suppurative inflammation of mastoids; laryngitis with necrosis	Edema, congestion, hemorrhage and collapse; early suppurative bronchitis	Focal glomerulitis	
Non-suppurative mastoiditis and laryngitis	Early mural bronchitis and mucous hypersecretion; emphysema	Thrombi in brain, thymus, larynx, and lung; focal adrenal hemorrhage; fatty degeneration of the liver	
Suppurative mastoiditis; tonsillitis with abscess; early suppurative laryngitis; non-suppurative tracheitis	Congestion, hemorrhage, edema and collapse	Thrombosis in brain and larynx; focal adrenal hemorrhage; testicular lymphocytic infiltration	Mastoid, trachea and lung: hemolytic *Staph. aureus*
Tonsillitis; non-suppurative inflammation of mastoids, larynx and trachea	Focal interstitial pneumonia; emphysema	Cardiac mural thrombi; submaxillary lymphocytic infiltration	
Suppurative mastoiditis; tonsillitis, early suppurative laryngitis, non-suppurative tracheitis	Focal interstitial pneumonia; edema, collapse, congestion and hemorrhage; bronchitis	Thrombosis in lung; advanced glomerulitis; adrenal hemorrhage; lymphocytic infiltration of testis	Heart's blood, mastoid, tonsil, and trachea: hemolytic *Staph. aureus*
Non-suppurative mastoiditis, laryngitis and tracheitis; tonsillitis	Edema, collapse, congestion, hemorrhage; bronchitis with early suppuration	Thrombosis in choroid plexus	Heart's blood, pericardial fluid, and mastoid: hemolytic *Staph. aureus*
Tonsillitis, laryngitis with early necrosis	Focal interstitial pneumonia; congestion, edema, hemorrhage and collapse; mural bronchitis	Glomerulitis; focal adrenal hemorrhage; lymphocytic infiltration of submaxillary gland	Heart's blood, sterile

TABLE I (Continued)

Case	Age and sex	Position when found	Exposure to respiratory disease	Prior history	Gross findings
No. 25 A 42 50	5 mos. Male	On abdomen, in crib, face to side	Negative	Cough for 2 weeks; had multiple transfusions for erythroblastosis	Normal mastoids; lungs: congestion and edema
No. 26 A 42 58	4 mos. Male	On abdomen, face down, in crib	Negative	None	Normal mastoid; lungs: 150 gm., edema and congestion
No. 27 A 42 80	1 mo. Male	On abdomen, face to side, in crib, bloody fluid on face	Positive	Was "getting" a cold	Normal mastoid; lungs: congestion and edema; uvula: granular
No. 28 A 42 99	3 mos. Male	Head against crib bumper; vomitus on sheet	Negative	History of allergy	Mastoids: "moist"; lungs: 100 gm., congestion and edema; bronchi contained blood-tinged mucoid material
No. 29 A 43 08	1 mo. Male	On back, in crib	Negative	Mother thought he had a cold	Mucoid exudate in mastoids; lungs: congestion and edema; diaper dermatitis. (Boric acid 0.4 mg. in 15.0 gm. of formalin-fixed liver and kidney)
No. 30 A 43 16	9½ mos. Male	On abdomen, in carriage	Negative	"Indigestion" the day before; vaccinated 3 days previously	Normal mastoid; lungs: 150 gm., emphysema, congestion and edema
No. 31 A 43 35	2½ mos. Male	In carriage, head under covers, on back	Mumps and chicken pox contact	None	Serous exudate in mastoids; lungs: 150 gm., congestion and edema; bronchi contained bloody material

clothes, and then only loosely. In the great majority of instances the face was turned to one side, permitting free access of air; a smaller group was comprised of those found lying on the back. Regardless of the position in which the infant was found, the pathologic findings were similar.

Routine microscopic studies have disclosed lesions that we believe provide an explanation for the sudden death. The significance of these lesions was appreciated only after the accumulation of much experience in the study of cases of this type along with that of control material. Our control material is of two kinds. We have made parallel histologic studies on infants dying after known violence,[2] as well as on infants *observed to die*[6] suddenly while in apparent health, or dying after a fulminating illness.[7] The two groups of cases we regard as

Microscopic findings			Bacteriologic findings
Upper respiratory tract	Lung	Other significant findings	
Non-suppurative laryngitis	Hemorrhage, collapse, edema and congestion; mural bronchitis	Lymphocytic infiltration of testis; focal adrenal hemorrhage	
Normal mastoid; non-suppurative laryngitis and tracheitis	Focal interstitial pneumonia; congestion, edema, hemorrhage and collapse	Glomerulitis; focal adrenal hemorrhage; thrombosis in cervical cord and lung; focal lymphocytic infiltration of testis and epicardium; fibrinoid necrosis in heart valve	
Suppurative mastoiditis; non-suppurative laryngitis and tracheitis	Congestion, hemorrhage, collapse and edema	Thrombosis in larynx and testis; focal adrenal hemorrhage; epicardial cellular infiltration	Spleen and lung, sterile
Non-suppurative laryngitis and tracheitis	Hemorrhage, congestion, collapse and edema	Thrombosis in choroid, thymus, larynx, lung and heart; glomerulitis; focal adrenal hemorrhage; focal epicardial infiltration	No pathogens
Non-suppurative inflammation of mastoids	Congestion, collapse, edema and hemorrhage	Thrombosis in lung; glomerulitis; pancreatic degeneration	
Normal mastoids; non-suppurative laryngitis	Emphysema; mural bronchitis with mucous hypersecretion	Thrombosis in lung and brain; focal adrenal hemorrhage; glomerulitis	No pathogens
Tracheitis; early suppurative laryngitis with necrosis	Focal lung abscess; collapse, congestion, edema; mural bronchitis	Thrombosis in lungs and larynx	Larynx, trachea, bronchus and liver: hemolytic *Staph. aureus* (*Staph. aureus* and *Strep. haemolyticus* in throat cultures of siblings)

"negative" and "positive" controls, respectively, for the microscopic changes which are reported in this paper.

Gross Observations at Necropsy

The 31 infants under discussion were well developed and well nourished. Hypostatic discoloration indicated the portion of the body that had been most dependent. This discoloration had usually shifted by change in position between the time of discovery of the body and the performance of the necropsy.

On opening the body cavities excess fluid was frequently seen in the pericardial sac and rarely in the pleural cavity. The heart's blood was usually completely or largely fluid and the right heart was more distended than the left. The thymus was of normal size for subjects of

this age. Petechiae were conspicuous in the thymus in 25 cases. They were often present in the pleura and pericardium.

Pulmonary congestion and edema were the most conspicuous gross changes (Fig. 1) and were present in all cases except 3; these showed emphysema. The pleural and cut surfaces were usually dark red to purple. The lung parenchyma was firm and rubbery, in contrast to the crepitant sensation imparted by the normal infant lung, which is pale pink-tan and soft (Fig. 2). A considerable amount of frothy fluid, often bloody, oozed from the cut surface of the lungs (Fig. 25).

The mucosa of the larynx, trachea, and bronchi was congested in one half of the cases. In about one third of the cases gray mucoid exudate was found in the lumina of the airway. The tracheobronchial and cervical lymph nodes usually were hyperemic and occasionally hemorrhagic. The tonsils seldom showed any gross changes. Mucoid material occasionally was seen in the nasopharynx. Non-occlusive amounts of regurgitated stomach contents were seen in the nasopharynx and larynx in 5 cases. Massive aspiration into the tracheobronchial tree occurred in only one case. As previously noted, we regard such an occurrence as probably agonal and of very little significance as a cause of death.

The stomach usually contained curdled milk. The lymphoid tissue of the bowel was prominent, as were the mesenteric glands. These were occasionally hyperemic. The liver and kidneys were frequently congested. The spleen was soft; and on section the follicular borders were indistinct. Rarely, the adrenal glands showed small gross hemorrhages.

On cisternal tap the cerebrospinal fluid often was found to be under increased pressure, the fluid frequently escaping several inches beyond the tip of the needle. The meninges usually were congested; often showed excess subarachnoid fluid; occasionally minute focal subarachnoid hemorrhages were seen. The middle ear and mastoid antra contained gross exudate in 17 of the 31 cases. In 6 cases this was purulent and in 11, either mucoid or serous.

Microscopic Observations

The changes found may be divided into two groups: local, involving mainly the respiratory tract, and systemic, involving other organs and the lymphatic tissues.

RESPIRATORY TRACT

Lungs

In 28 of the 31 cases, diminution in the alveolar air space was the most conspicuous change in the lungs. This was the result of pulmo-

nary congestion, edema, hemorrhage, collapse, and cellular infiltration (Figs. 3, 9, 14, 28, and 32). The extent of each of these changes varied considerably.

Congestion was present in the pulmonary and bronchial blood vessels and was mainly venous and capillary. The red cells were closely packed. Clumps of granular eosinophilic material, usually interpreted as representing platelets, were numerous; and in 14 cases actual pulmonary thrombi were recognized in routine sections (Figs. 3 and 31); these contained fibrin, "platelets," leukocytes, and erythrocytes in varying proportions. In several instances the vessel wall at the site of thrombosis was infiltrated by mononuclear cells, among which there were large cells with acidophilic cytoplasm. Mural edema was frequent, and degeneration of the elastic fibers was seen occasionally.

The alveolar capillaries often were extremely engorged (Figs. 9, 32, 46, and 48). Interstitial and alveolar hemorrhages occurred in all but 6 of the 31 cases (Fig. 10). In some instances the interstices were transformed into pools of blood, and whole pulmonary lobules appeared to be infarcted. Vessels were often surrounded by sheaths of blood (Fig. 14); and the alveolar capillaries occasionally appeared to be disintegrating.

The pulmonary congestion was usually associated with varying degrees of edema, both interstitial and intra-alveolar. The interlobular septa frequently were converted into broad pink bands (Fig. 29). The blood vessels and bronchial walls often were thickened by edema and distended interstitial lymphatics often contained leukocytes, erythrocytes, and cell débris. Intra-alveolar edema and subpleural edema frequently were marked.

Pulmonary collapse was prominent. In some instances it seemed to be the result of occlusion of bronchi and bronchioles by mucoid secretion and desquamated and degenerated epithelium. In others, the collapse was associated only with congestion, edema, and hemorrhage. The alveolar air space often was reduced further by the presence of alveolar macrophages (Figs. 46 and 48); in 6 of the 31 cases alveolar giant cells were conspicuous. In 7, there was erythrocytophagocytosis by alveolar macrophages.

In 7 cases there were foci of polymorphonuclear leukocytes in the alveolar lumina (Figs. 7 and 8) and in 5 others there were foci of interstitial and intra-alveolar mononuclear infiltration (Figs. 5 and 6). Thus, 12 of the 31 cases showed either focal bronchopneumonia or focal interstitial pneumonitis (microscopic) in the sections routinely examined. In 11 other cases there was slight lymphocytic infiltration confined to some interlobular septa. In one case there was a minute

abscess, 3 mm. in diameter, discovered in one of the sections taken for microscopic study.

There was some degree of bronchitis or bronchiolitis in every case (Figs. 11, 12, and 13). This was mural in 23 and only catarrhal in 8. Epithelial proliferation, degeneration, and desquamation, mural hyperemia, edema, and cellular infiltration were prominent. In the large bronchi there often were hypersecretion and degeneration of the mucous glands. Not all bronchi or bronchioles in each section were involved; and in several cases the extent of this change was limited. In 15 cases epithelial proliferation, degeneration, and desquamation were sufficiently marked to occlude the lumina (Fig. 28). This material constituted the gray mucoid exudate noted grossly. Erythrocytes and mononuclear cells usually were admixed with the epithelial débris. In 10 instances there were also a few polymorphonuclear leukocytes within the lumina (Figs. 33 and 34). Denudation of epithelium often was extensive, with only fragments of the basal layer remaining. Occasionally, granular eosinophilic precipitate and leukocytes were seen between the basement membrane and the desquamating and degenerating epithelium. The mural cellular infiltration was mainly lymphocytic; large mononuclear cells with acidophilic cytoplasm and plasma cells occasionally were prominent. In 5 instances polymorphonuclear leukocytes were present also within the bronchial wall, and, rarely, eosinophilic granulocytes.

The pleura often showed hyperemia, edema, and focal hemorrhages; occasionally focal mononuclear cellular infiltrate was noted.

The bronchopulmonary lymph nodes showed hyperemia and edema. The normal follicular pattern was lost. Diffuse hyperplasia or lymphoid depletion was encountered. Sinus catarrh and erythrophagocytosis were frequent.

Larynx and Trachea

In 9 cases foci of necrosis were found in the larynx and trachea. In 2 cases this was deep within the larynx, affecting muscle or mucous gland (Fig. 17). In 7 cases it was found in the vocal cord (Figs. 15 and 16). The earliest lesion seen in the vocal cord consisted of vacuolar degeneration of the stratified squamous epithelium and fibrinoid change in the connective tissue of the vocal ligament (Fig. 36). In more advanced lesions, edema, hyperemia, and cellular infiltration appeared. The capillary endothelial lining was swollen. The cellular infiltrate consisted of mononuclear cells, many of which were pyknotic; polymorphonuclear cells were seen in the areas where necrosis was more marked.

In 8 additional cases there was focal or diffuse polymorphonuclear infiltration of larynx or trachea, unassociated with necrosis in the sections studied.

In most cases there was widespread injury of the laryngeal or tracheal epithelium, consisting of degeneration with loss of cilia, desquamation, and proliferation. Spaces distended with lymphocytes or polymorphonuclear leukocytes frequently were found within the epithelium. Desquamating epithelium sometimes was separated by granular precipitate and leukocytes from the substantia propria. The mucous glands often showed striking degeneration of their epithelium and distention of the acinar and duct lumina with epithelial débris, red blood cells, and leukocytes. The glands were frequently surrounded by lymphocytes and plasma cells.

Tonsil and Nasopharynx

Of the 16 cases in which sections of the tonsil were available, polymorphonuclear leukocytes were found within the crypts and infiltrating the epithelium in 12 (Figs. 18 and 19). In the subjacent connective tissue the lymphatics usually were distended with lymphocytes, many of which were pyknotic. Perivascular mononuclear cellular infiltration, mural vascular edema, and thrombosis were seen occasionally. The striated muscle frequently showed interstitial edema and occasionally waxy degeneration of the fibers. In 2 cases of the few in which pharyngeal mucous membrane had been sectioned a striking lesion was found in the form of marked edema of the submucosa and muscle with extensive cellular infiltrate, both mononuclear and polymorphonuclear (Figs. 40 and 41).

Middle Ear and Mastoid Antra

As noted previously, in 17 of the 31 cases there was gross exudate in the middle ear and mastoid antra. In 6 instances this was purulent (Fig. 39); and in 11 either mucoid or serous. In those showing grossly only mucoid or serous exudate, polymorphonuclear leukocytes were found microscopically in 5 instances (Fig. 38), but all had mononuclear infiltration. Of the 14 mastoids which were normal grossly, sections were available in 10. In 8 of these there was cellular infiltration; in 3 this was largely polymorphonuclear; and in 5 mononuclear with occasional eosinophils.

Thrombi were found in sections of the upper respiratory tract (tonsil, pharynx, and larynx) in 8 instances of this series.

OTHER ORGANS

Central Nervous System

In 21 cases the meninges were edematous. In 18 there was increased cellularity; the infiltrating cells were lymphocytes and large mononuclear cells with acidophilic cytoplasm. Focal subarachnoid hemorrhages were found in 15 cases, and were associated with intense vascular engorgement and meningeal edema. Perivascular hemorrhages were found frequently in the brain substance, and here also were associated with intense vascular engorgement and mural vascular edema. Thrombi were seen in the meninges, brain, or choroid plexus in 11 cases.

Endocrine Glands

In every instance the pituitary, thyroid, and adrenal glands were hyperemic. Focal adrenal hemorrhages were present in 20 cases; thrombosis in addition to adrenal hemorrhage was seen once (Fig. 21). Thrombosis in the pituitary gland also was seen once in this series. The thyroid gland usually showed depletion of colloid, collapse of vesicles, and filling of the lumina with desquamated or proliferated epithelium.

Heart

In 3 cases there were foci of perivascular lymphocytic infiltration in the epicardium (Fig. 23). In 2 there was fibrinoid necrosis of the mitral valve and in one there was coronary sclerosis. Hyperemia and interstitial edema were noted frequently in the heart muscle.

Liver

In 17 cases there were periportal or parenchymal foci of lymphocytic infiltration in the liver (Fig. 22). These usually were small, but in 2 instances the reaction was quite marked; pyknosis of lymphocytes often was seen within these foci. There usually was congestion of the sinusoids and in 2 instances a diffuse fatty degenerative change was seen (Fig. 34).

Salivary Gland and Testis

Lymphocytic infiltrate was present in 7 of the 11 submaxillary glands available for study. In this series there were 20 males; sections of testes were taken in 17 instances. Testicular lymphocytic infiltration was seen in 9 cases. Two cases showed both submaxillary and testicular infiltration. Lymphocytes, large mononuclear cells, and occasionally plasma cells were seen (Figs. 49 and 50).

Kidney

In the kidneys of 6 cases there was focal glomerulitis with congestion of capillaries, proliferation of endothelium, and occasionally fibrin thrombi or fibrinoid necrosis. In one kidney this change was extensive (Fig. 24). The occasional fibrotic glomerulus seen in 6 other cases was not considered significant in view of the frequency of this finding in our "normal control" material.[2]

LYMPHATIC TISSUES

The most conspicuous change in lymphatic tissues was hyperacute involution of the thymus, as indicated by hyperemia, hemorrhage, thrombosis, and destruction of lymphocytes with accumulation of abundant cytoplasmic and nuclear débris in enlarged Hassall's corpuscles.

The lymph nodes lost their nodular architecture and their normally developing germinal centers. The reticular cells showed increased phagocytic activity. There was either diffuse hyperplasia or depletion of lymphocytes. Plasma cells sometimes were numerous; polymorphonuclear leukocytes and eosinophils occasionally were conspicuous. Hyperemia and focal hemorrhages were prominent (Fig. 20). While the involvement was most intense in the tracheobronchial and cervical groups, peripheral and mesenteric nodes also were affected.

In the spleen distinct demarcation between the white and red pulp usually was lost. The malpighian bodies no longer showed a distinct follicular border (Fig. 42) and were often reduced in number and size; they seldom showed germinal centers, and occasionally pronounced reaction centers were seen. Hemosiderin occasionally was conspicuous. Pyknosis of lymphocytes was a common finding in all of the lymphatic tissues.

ETIOLOGIC STUDIES

Bacteriologic studies were carried out in 24 of the 31 cases. Mixed flora of questionable significance were frequent. In 11 instances hemolytic *Staphylococcus aureus,* and in 3, *Streptococcus haemolyticus,* were recovered from the organs at necropsy. The tracheobronchial tree and mastoid were the usual sites from which these organisms were recovered. In one case a pure culture of hemolytic *Staph. aureus* was recovered from the heart's blood, lung, and spleen. These organisms have been recovered occasionally from infants dying immediately after violence;[2] therefore their significance, except as secondary invaders, is questionable. An attempt to isolate viral agents is being conducted

by Dr. Karl Habel at the National Institute of Health in connection with the coordinated study of the problem of sudden death during infancy now being made at four centers.*

Toxicologic Studies

Early in the course of the study of this problem the organs of many of the cases were submitted to Dr. Alexander O. Gettler, Toxicologist of the Office of the Chief Medical Examiner, City of New York, for examination for known poisons. These results were negative. However, in one case of this series in 1950 there was present 0.4 mg. of boric acid in 15.0 gm. of fixed liver and kidney; and in another, 0.15 mg. in 12.0 gm. of tissue. These amounts are not considered significant. The discovery of a lethal amount (210 mg. in 200 gm. of fresh liver and kidney) of boric acid in another case of unexpected death (not found dead) emphasized the necessity of testing for this poisonous agent in all cases that show a "diaper" dermatitis.

Representative Cases

Case 6 (ME 50–28)

According to the mother, this 9-months-old infant had had a cold for several days and had breathed heavily the night before. He was last seen alive about 5 a.m. when he was given a bottle. About 10 a.m. he was found dead with his head at the foot of the carriage, covered by a house dress. Although he had often twisted around and slept in that position, the mother was sure that he had suffocated. He had a crusted lesion behind the ears that had been diagnosed as "allergic" by a health station physician.

At necropsy there was mucoid material in both middle ears and mastoid antra, and congestion of the cerebral vessels; a very small amount of aspirated stomach contents was seen extending into the upper trachea and there was congestion and edema of the lungs.

Microscopic Findings. In the lung section there was marked collapse and congestion. Interstitial and intra-alveolar edema and hemorrhage were moderately conspicuous. In one section only, a minute focus of intra-alveolar polymorphonuclear leukocytic exudation was seen. Intra-alveolar macrophages were prominent. In the bronchi there was degeneration and desquamation of epithelial cells and mucous hypersecretion. There was marked mural infiltration with mononuclear cells; a few polymorphonuclear leukocytes also were seen in the bronchial walls and lumina. In the epithelium lining the mastoid cells there was edema and infiltration by mononuclear cells and poly-

* Supported by a grant from the Federal Security Agency, United States Public Health Service.

morphonuclear leukocytes (Fig. 38). In the tonsil there was hyperemia; areas of the epithelium were undergoing necrosis and there was also polymorphonuclear leukocytic infiltration. In the larynx and trachea there was degeneration of the epithelium, conspicuous subepithelial mononuclear infiltration, and marked degenerative changes in the submucous glands. Focal hemorrhages and diffusion of lymphocytes were seen in the cervical lymph nodes and spleen. In the kidney there was advanced glomerulonephritis (Fig. 24). In the liver there were a few foci of mononuclear infiltration. In the pancreas there was interstitial edema. In the adrenal gland there was focal hemorrhage. Thrombi were seen in the mesenteric vessels. The cerebral vessels were hyperemic and there was an occasional focal hemorrhage.

Case 8 (ME 50–47)

According to the mother, this 3-weeks-old female infant had been entirely well. She was found dead 3½ hours after her last feeding. She was lying on her back, head not covered; there was no vomitus on the face or bedclothes. The day after this infant's death an older sibling developed a sore throat.

At necropsy the mastoids were normal grossly. There was no aspiration of stomach contents. Petechiae were prominent in the thymus, epicardium, and pleura. The lungs were extremely dark, purplish red and moderately edematous. The spleen was soft; the follicles indistinct.

Microscopic Findings. In the lungs there was extremely marked collapse with congestion. There was moderate intra-alveolar edema. In several large pulmonary veins there were thrombi (Fig. 3). The vessel walls at the sites of thrombosis had focal infiltration by eosinophilic mononuclear cells, plasma cells, and a few eosinophils. The bronchial walls showed edema, but no infiltration. There was moderate epithelial degeneration and desquamation. In the tracheal lumen there was a small amount of mucoid material mixed with lymphocytes and a few polymorphonuclear cells. The cervical lymph nodes were hyperemic. The mastoid epithelium showed infiltration by eosinophils and polymorphonuclear leukocytes. There were focal adrenal hemorrhages. In the meninges there were focal perivascular hemorrhages.

Culture of the lung was sterile; no pathogenic organisms were recovered from the other viscera.

Case 15 (ME 50–133)

According to the mother, this 5-day-old infant had been discharged from the hospital on the day of death. The hospital record revealed that the infant's progress was believed to have been entirely normal. He was found dead in his crib face down ½ hour after a feeding. There was no vomitus on the bedclothes. The infant and

mother were Rh positive and the direct Coombs' test of post-mortem blood was negative. A sibling had a cold.

At necropsy the mastoids were normal. There was no aspiration of stomach contents. The lungs showed moderate congestion and edema with a few pleural petechiae. The spleen was soft and had indistinct follicles. The thymus was markedly involuted.

Microscopic Findings. In the lung there was extreme loss of aeration as the result of collapse, congestion, and edema. A moderate number of squames (from vernix) were seen in some alveoli. A single small focus of intra-alveolar lymphocytic and polymorphonuclear leukocytic exudation was seen in one section. In the bronchi, edema of the walls was conspicuous but there was no infiltration. In a vocal cord there was focal necrosis with infiltration by pyknotic lymphocytes and polymorphonuclear leukocytes (Fig. 16). There was ulceration of the overlying epithelium. In the myocardium a focus of mononuclear infiltration was seen. Similar foci, mainly perivascular, were seen also in the testis and submaxillary gland. Thrombi were seen in the vessels of the brain, thymus, and lung. There was interstitial edema in the pancreas.

Discussion

In evaluating the significance of morphologic findings in infants found dead the lesions were compared with those in infants dying of violence. In those found dead morphologic evidence indicating acute respiratory disease was present in all infants; the lesions were multiple, although they varied as to their location and also as to their extent. In the group with violent deaths,[2] local lesions in the respiratory tract were not constant, usually not multiple, and when present were mild. One would expect to find minor lesions, acute or not, in the respiratory tract of some infants regardless of the cause of death, since respiratory infections are so common in infancy and early childhood.

An equally significant difference was found in these two series of cases with reference to systemic changes in the blood vessels and the lymphatic system. In not one of the examples of violent death was there loss of the normal lymph-follicular pattern or the extent of depletion of lymphocytes or hyperplasia seen in the infants found dead. In no case of death immediately after trauma was there found the same degree of hyperemia or hemorrhage in the thymus, bronchopulmonary and cervical lymph nodes. Also absent from these cases was the extreme visceral congestion. Even in death by strangulation, the viscera did not show the petechiae that were so prominent in most

infants found dead. Thrombosis, which was found in 20 of the 31 cases on routine section, was never seen in those dying of trauma.

The vascular changes and lymphatic tissue reactions are, in a sense, non-specific. They occur in various infections, such as meningococcemia and pneumonia, in any age group, and can be induced readily in the experimental animal, not only by infectious agents, but also with non-living antigens such as bacterial vaccines.[8-10]

In the past 20 years we have studied also many cases of infant deaths reported to the Medical Examiner's Office because death occurred after a fulminating clinical illness lasting, in most cases, from a few minutes to a few hours.[7] The gross necropsy findings in some were inconclusive; the microscopic changes were analogous to those encountered in infants found dead. In others, a conventional gross cause of death was found, such as bronchopneumonia, laryngotracheobronchitis, or meningitis. The same vascular changes described in infants found dead were often prominent.

One is tempted to suggest, therefore, that the vascular changes common to all groups of infants under discussion may represent an important, perhaps not less important, factor in the pathogenesis of the fatal disease than the local lesion which is ordinarily accepted as a conventional cause of death.

We have previously shown that sudden, apparently unexplained death during early life rarely occurs after the age of 1 year and is mainly a problem of the first 6 months of life.[3-5] This is the period when the prognosis in pneumonia is gravest.[11] We have shown also that in New York City the seasonal incidence of deaths previously ascribed to accidental mechanical suffocation parallels those certified as being due to respiratory infections.[3] Despite the original report that these infants were apparently healthy, careful investigation at the scene of death and in follow-up interviews with the parents often revealed a history of exposure to respiratory disease. In many, a history of a recent cold or of other symptoms referable to respiratory disease was obtained also. We have had instances in which one of twins was found dead and the other subsequently became ill of respiratory disease. At the present time the etiologic agents responsible for the great majority of respiratory diseases are unknown. It is not surprising, therefore, that the specific causes for the fulminating respiratory disease in the infants found dead remain to be discovered.

Until very recently smothering has been the traditional explanation for the sudden deaths of infants while in apparent health. However, as early as 57 years ago Brouardel and Benham,[12] under the heading

of pulmonary congestion, called attention to bronchitis as a common cause of sudden deaths in infants "5 or 6 months old." "A child is found dead in its cradle one morning, and its nurse and mother, especially if the mother is an unmarried girl, are accused of having smothered the child by pressure with the arm. An autopsy is ordered, and this is what is usually found:—You know that, in what is called 'capillary bronchitis' or 'suffocating catarrh of children' (Laennec), intense pulmonary congestion now and then occurs, which places the child's life in jeopardy for several hours. When the child is strong, and is more than 7 or 8 months old, it seldom dies of the first attack of congestion. But when the age is less than 6 months, death may occur in the first attack ....." Unfortunately, their paper attracted little, if any, interest. Farber,[13] in 1934, called attention to fulminating streptococcal septicemia as a cause of sudden death in infants. He reported 2 cases in which the infants were found dead, allegedly of suffocation, with streptococci in the heart's blood and with early interstitial infiltration of the alveolar walls and peribronchial lesions.

In 1938 Goldbloom and Wiglesworth,[14] in Canada, presented evidence of respiratory infection sufficient to explain 19 of 30 sudden deaths in infants. In 1942 one of us (J. W.)[15] reported upon the association of histologic evidence for fulminating respiratory disease with sudden, apparently unexplained death during infancy. In 1945 Davison[4] noted a precipitous drop in the incidence of mechanical suffocation in his jurisdiction as a result of the routine performance of necropsies on infants found dead. Of 318 cases investigated by him, he concluded that suffocation was present in only 24. In a series of 167 cases reported by us in 1947,[3] we found no evidence to support the conclusion that smothering had occurred; the great majority showed lesions of the respiratory tract and other organs.

In 1944 Abramson[16] reported an increasing number of infant deaths certified as due to accidental mechanical suffocation. An analysis of a representative sample of the cases reported by him shows that in the majority necropsies were not performed, and in those that were necropsied many did not have an examination of the brain, neck organs, middle ears, or mastoids; and, with one exception, microscopic findings were not recorded. We[3] showed that in the New York City area the increasing incidence of death due to "accidental mechanical suffocation" reflected a corresponding decrease in deaths certified as status thymicolymphaticus. It was obvious, therefore, that one unsatisfactory "cause" of death was being substituted for another.

Bowden and French,[17] in their second series of unexpected deaths

in infants and young children, reported the finding of histologic evidence of tracheobronchitis and acute bronchitis in 29 of 43 cases. Gruenwald and Jacobi[18] reported the presence of "mononuclear pneumonia" in 47 of 52 consecutive cases of sudden rapid death in infants. Since mononuclear cells respond readily to any of a great variety of stimuli, including that of a protracted death agony (as seen in infants dying after known violence), we do not consider that they are significant by themselves. For the same reason the term giant cell pneumonia[19] is objectionable.

SUMMARY

Thirty-one consecutive cases of infants dying suddenly while in apparent good health and with gross necropsy findings insufficient to explain death were studied microscopically. In all cases there were microscopic inflammatory lesions in the upper and lower respiratory tracts. There were vascular changes both in the respiratory tract and in other organs. The spleen, lymph nodes, and thymus showed characteristic reactions. The presence of these changes may be regarded as evidence that death was caused by fulminating respiratory disease.

Acknowledgment is made to Miss Lillian Lesser for the photomicrographs.

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LEGENDS FOR FIGURES

FIG. 1. ME 42–36. Female, 2 months old. Found dead in crib, allegedly suffocated. Lung: Extreme congestion. For comparison with Figure 2.

FIG. 2. ME 42–65. Male, 8 months old. Death by drowning. Lung: Normal air content; absence of congestion.

FIG. 3. Case 8 (ME 50–47). Female, 3 weeks old. Found dead. Lung: Extreme collapse, congestion; thrombus in pulmonary vein. For comparison with Figure 4. × 12.

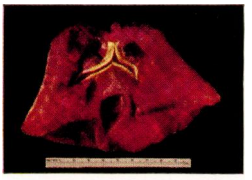
FIG. 4. ME 46–101. Male, 2½ months old. Death by drowning. Lung: Normal bronchi and alveoli; complete absence of congestion, edema, and infiltration. × 21.

FIG. 5. ME 44–309. Male, 3 months old. Found dead. Lung: Focal interstitial pneumonitis. × 34.

FIG. 6. High power view of a portion of Figure 5. × 146.

FIG. 7. Case 12 (ME 50–77). Male, 10 days old. Found dead. Lung: Focal bronchopneumonia. × 42.

FIG. 8. High power view of a portion of Figure 7. × 180.



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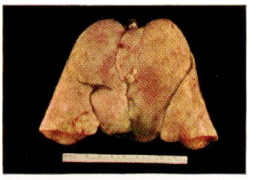

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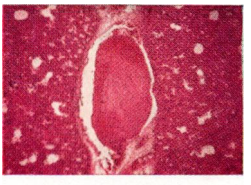

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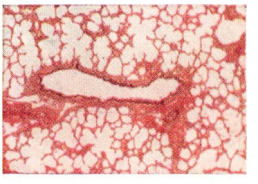

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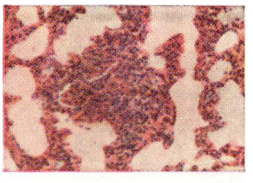

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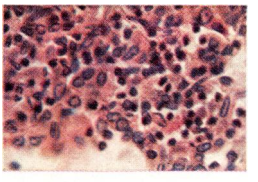

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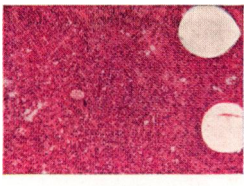

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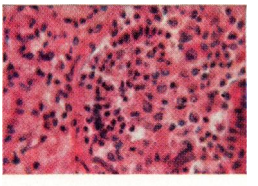

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FIG. 9. Case 11 (ME 50–74). Male, 6 weeks old. Found dead. Lung: Extreme capillary engorgement. × 70.

FIG. 10. ME 47–17. Male, 3 months old. Found dead. Lung: Interstitial edema and hemorrhage. × 65.

FIG. 11. Case 5 (ME 50–14). Female, 3 weeks old. Found dead. Lung: Mural bronchitis with hyperemia, edema, and mononuclear cellular infiltration. Of note is the alveolar capillary congestion. × 35.

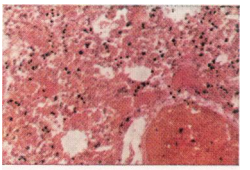
FIG. 12. High power view of a portion of Figure 11. × 145.

FIG. 13. Case 7 (ME 50–30). Male, 2 months old. Found dead. Lung: Bronchitis with epithelial degeneration and desquamation; a few polymorphonuclear leukocytes (seen in higher magnification) are admixed with the epithelial débris. × 35.

FIG. 14. Case 28 (A 4299). Male, 3 months old. Found dead. Lung: Perivascular hemorrhage. Of note also are pulmonary collapse and congestion. × 42.

FIG. 15. Case 18 (ME 50–143). Male, 1 month old. Found dead. Larynx through vocal cord: Degeneration of epithelium; edema and early necrosis of connective tissue with early infiltration by polymorphonuclear leukocytes and mononuclear cells. × 42.

FIG. 16. Case 15 (ME 50–133). Female, 5 days old. Found dead. Larynx through vocal cord: Necrosis with polymorphonuclear leukocytic and mononuclear cellular infiltration. × 70.



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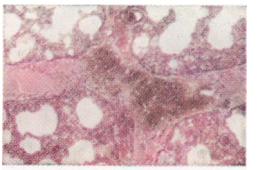

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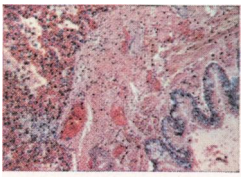

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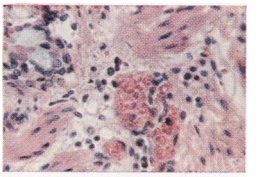

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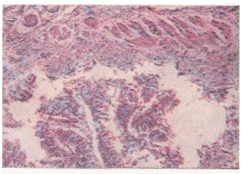

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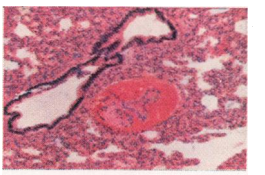

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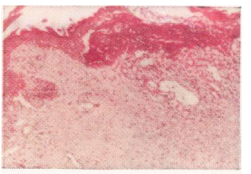

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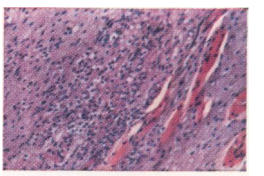

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FIG. 17. Case 20 (ME 50–168). Male, 2 months old. Found dead. Larynx: Mononuclear cellular infiltration of gland deep within larynx; exudate within duct. × 180.

FIG. 18. Case 20 (ME 50–168). Male, 2 months old. Found dead. Tonsil: Acute tonsillitis; polymorphonuclear leukocytic infiltration; pus in crypt. × 24.

FIG. 19. High power view of a portion of Figure 18. × 300.

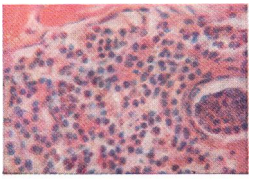
FIG. 20. Case 5 (ME 50–14). Female, 3 weeks old. Found dead. Cervical lymph node: Hyperemia and focal hemorrhage. × 12.

FIG. 21. Case 12 (ME 50–77). Male, 10 days old. Found dead. Adrenal gland: Thrombus in vessel and focal hemorrhage in inner cortical zone. × 42.

FIG. 22. Case 7 (ME 50–30). Male, 2 months old. Found dead. Liver: Periportal mononuclear cellular infiltration. × 145.

FIG. 23. Case 28 (A 4299). Male, 3 months old. Found dead. Heart: Perivascular lymphocytic infiltration. × 42.

FIG. 24. Case 6 (ME 50–28). Male, 9 months old. Found dead. Kidney: Azocarmine stain showing active glomerulonephritis with fibrinoid degeneration and fibrosis of glomerular capillaries. × 35.



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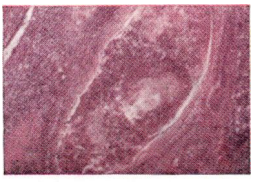

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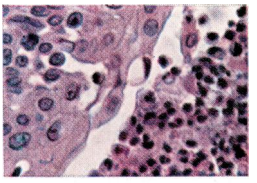

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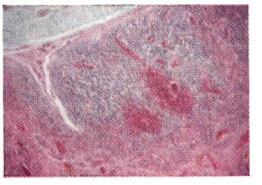

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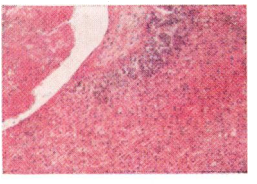

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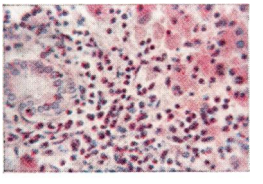

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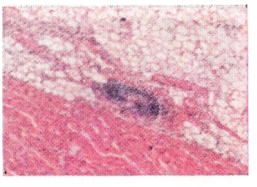

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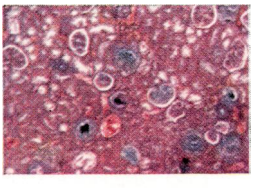

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FIG. 25. ME 42–36. Female, 2 months old. Found dead in crib, allegedly suffocated. Lung: Cut section showing extreme congestion and edema.

FIG. 26. ME 46–101. Male, 2½ months old. Death by drowning. Lung: Normal bronchi and vessels; well aerated normal alveoli found throughout the greater portion of the lung. × 40.

FIG. 27. Periphery of lung of same case as Figure 26, showing collapse. This change was limited to small areas in the periphery. There is no congestion. × 40.

FIG. 28. ME 52–94. Male, 13 days old. Found dead. Lung: Collapse, congestion, intra-alveolar edema, and macrophages. Of note also are the bronchial epithelial degeneration and desquamation with obstruction of the lumen. For comparison with Figures 26 and 27. × 40.

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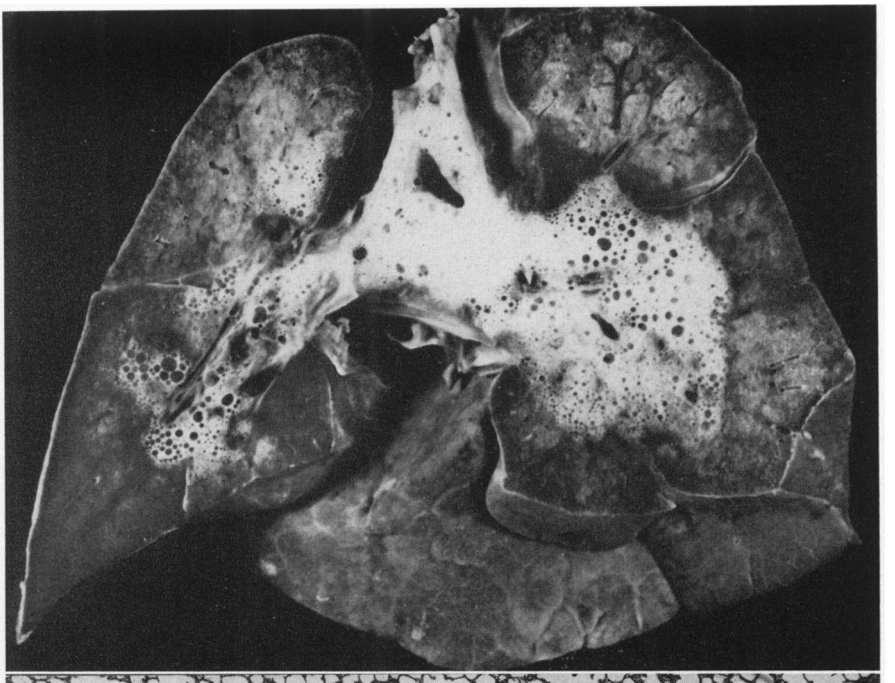

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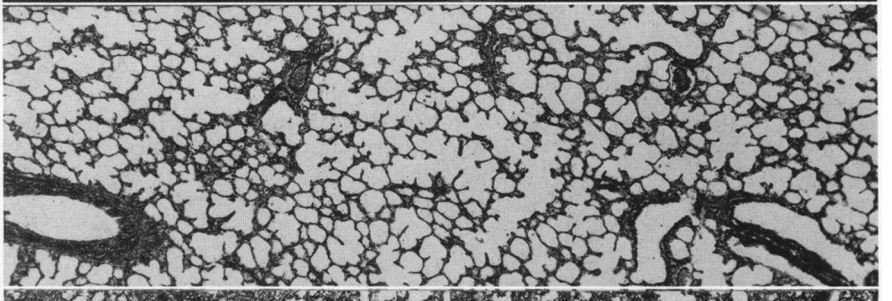

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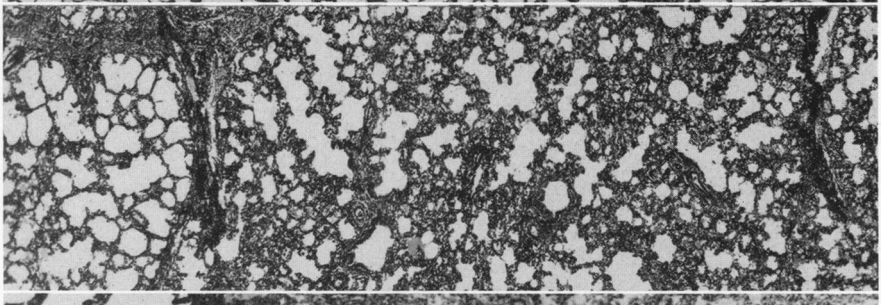

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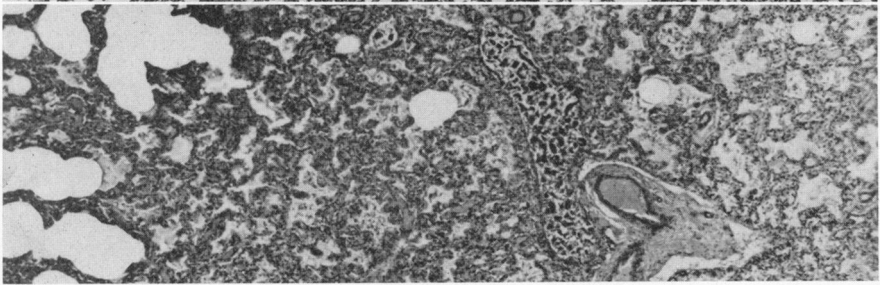
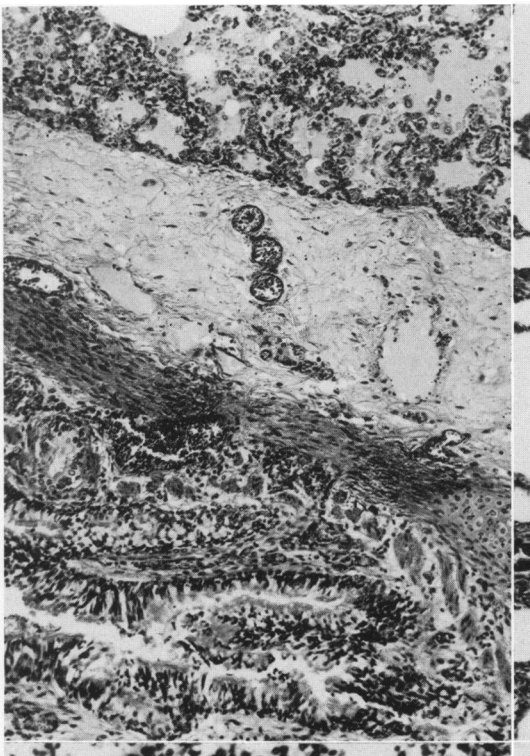


FIG. 29. Case 4 (ME 50–11). Female, 7 weeks old. Found dead. Lung: Interstitial edema, dilated lymphatics, and hyperemia; alveolar and bronchial hyperemia and edema. × 110.

FIG. 30. ME 46–101. Female, 2½ months old. Death by drowning. Normal lung: For comparison with Figure 32. There is complete absence of edema, congestion, and intra-alveolar macrophages. × 110.

FIG. 31. Case 19 (ME 50–159). Female, 3 months old. Found dead. Lung: "Platelet" and leukocytic thrombus in pulmonary vein. × 205.

FIG. 32. Case 5 (ME 50–14). Female, 3 weeks old. Found dead. Lung: Extreme alveolar capillary congestion; edema fluid and macrophages in alveolar lumina. × 110.



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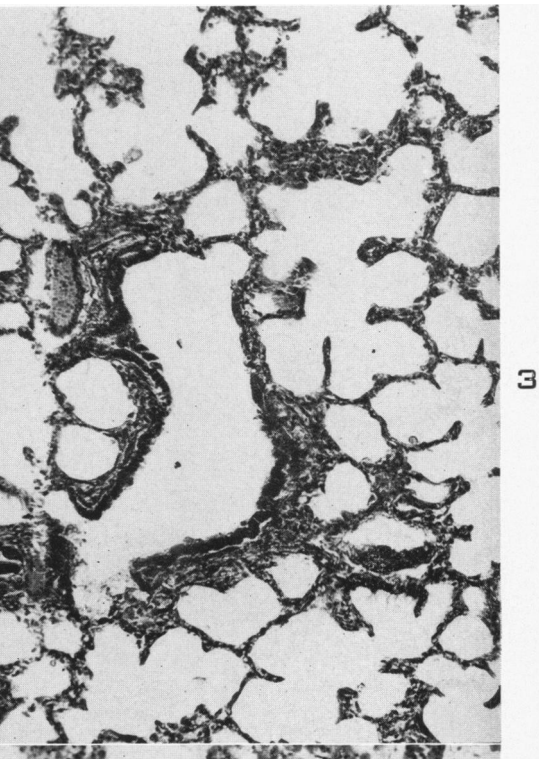

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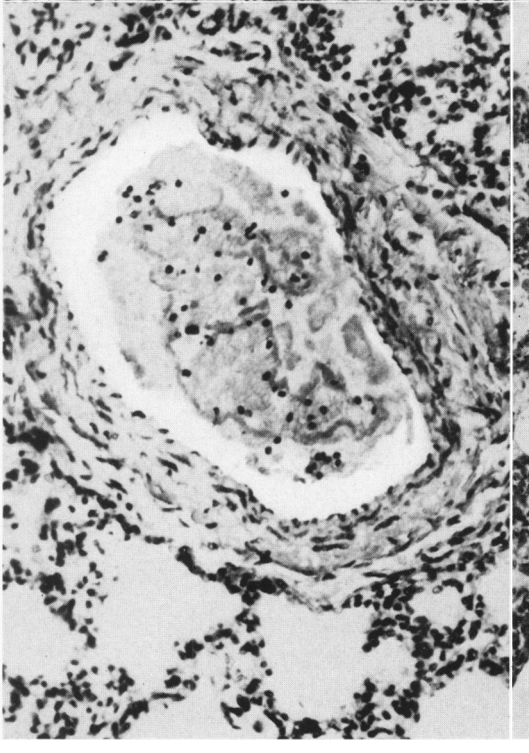

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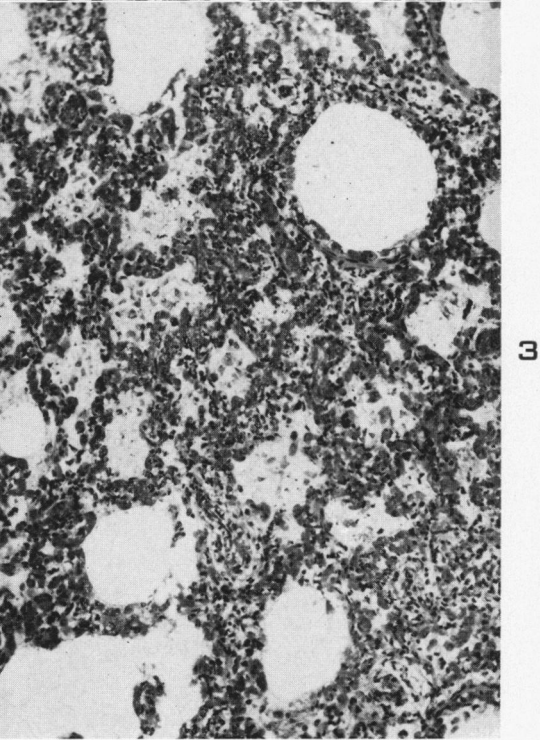


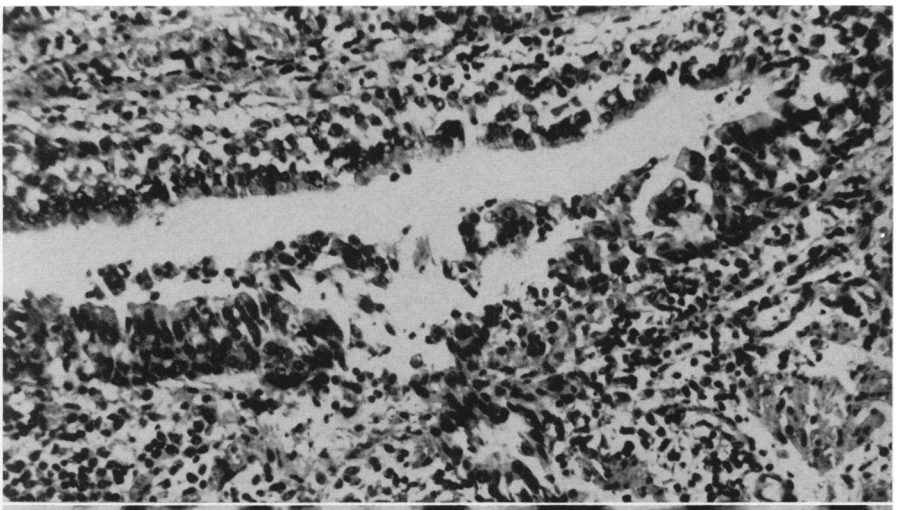
32

FIG. 33. 52–A–14. Female, 4 months old. Found dead. Bronchiole: Polymorphonuclear leukocytic infiltration and exudation. × 230.

FIG. 34. A portion of the area of bronchiolitis shown in Figure 33 at much higher magnification. × 978.

FIG. 35. Case 19 (ME 50–159). Female, 3 months old. Found dead. Liver: Fatty change. × 205.

33



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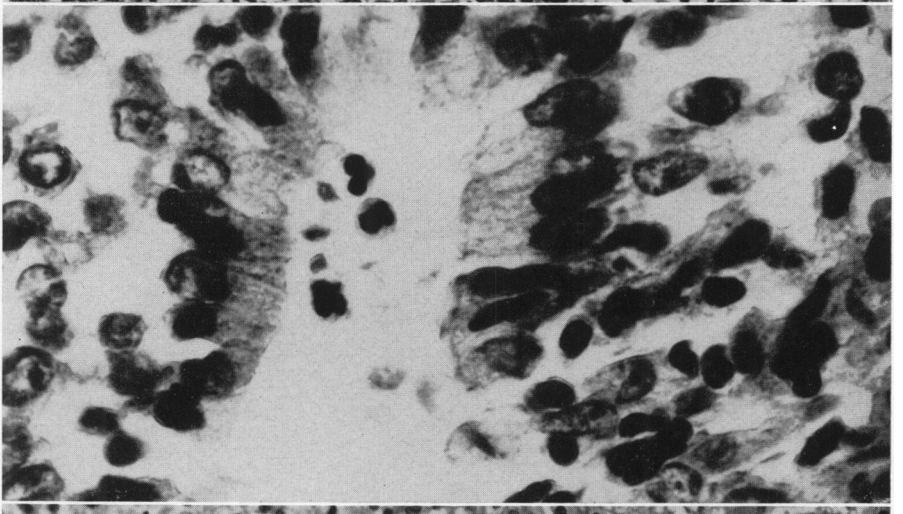

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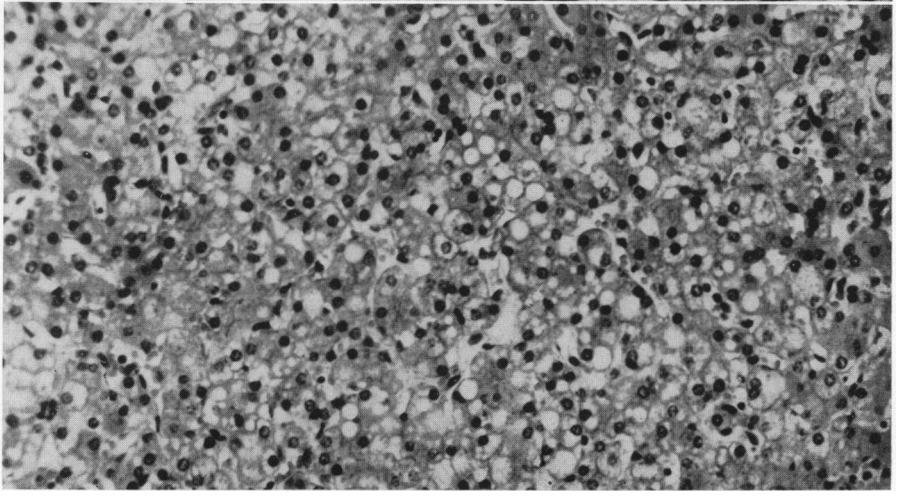
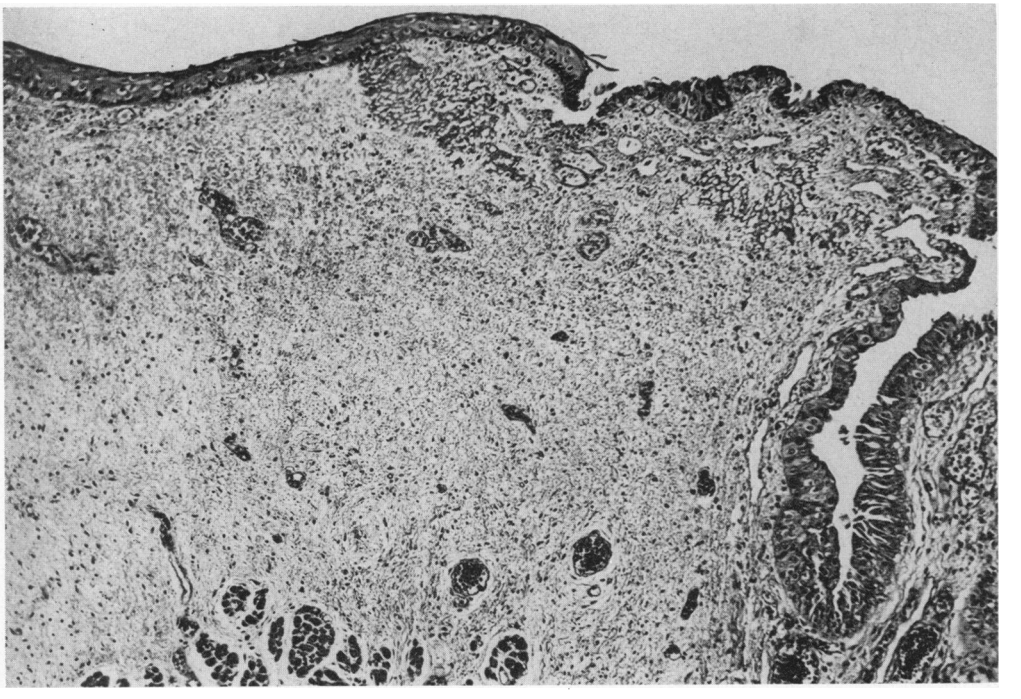


FIG. 36. Case 12 (ME 50–77). Male, 10 days old. Found dead. Larynx through vocal cord: Degeneration of the stratified squamous epithelium and fibrinoid change in vocal ligament. × 110.

FIG. 37. ME 52–145. Female, 4 years old. Homicidal strangulation. Mastoid: Normal mastoid with thin lining epithelium; there is no cellular infiltration. For comparison with Figures 38 and 39. × 66.

FIG. 38. Case 6 (ME 50–28). Male, 9 months old. Found dead. Mastoid: Moderate edema and infiltration of lining epithelium by mononuclear cells and polymorphonuclear leukocytes. Grossly, this mastoid contained mucoid material. × 66.

FIG. 39. Case 3 (50–A–54). Male, 6 weeks old. Found dead. Mastoid: Edema and infiltration by polymorphonuclear leukocytes and mononuclear cells. This mastoid was grossly purulent. × 66.



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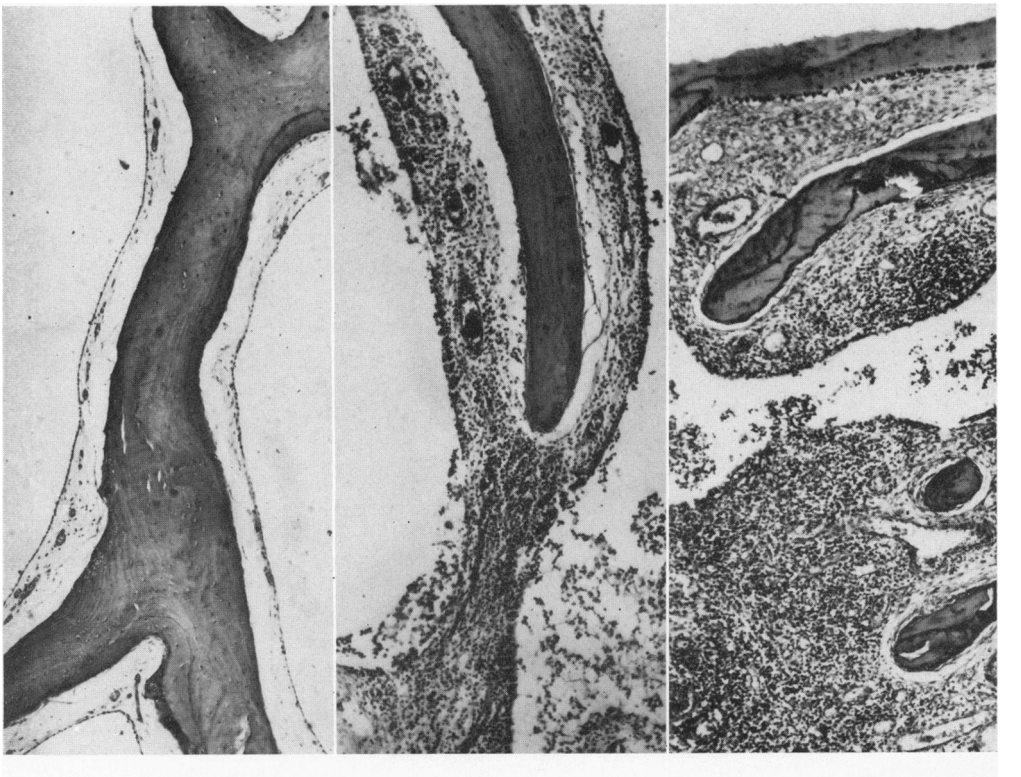
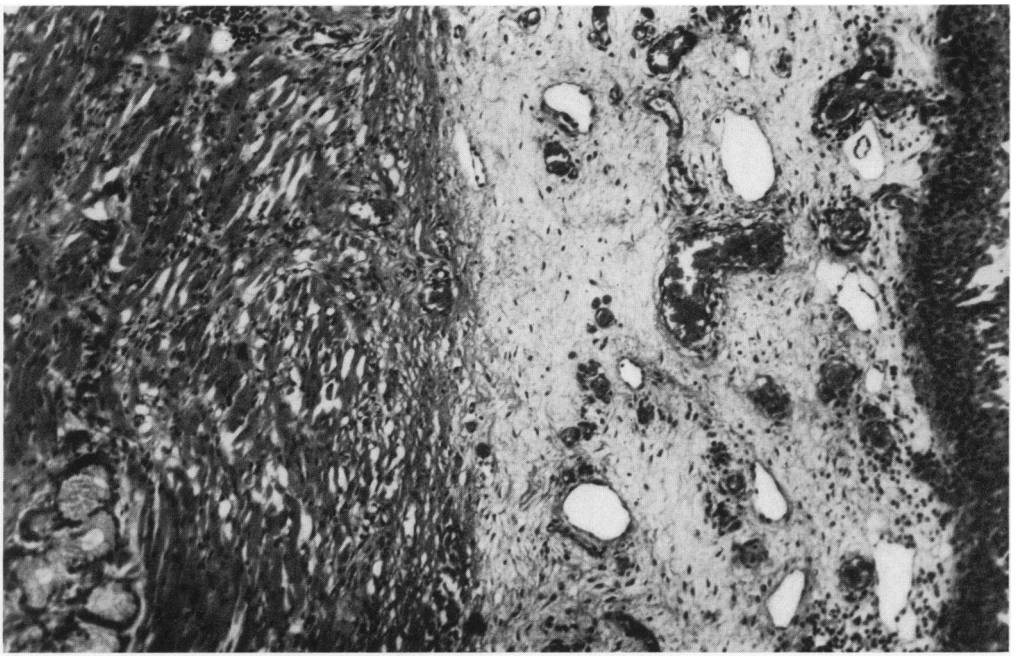


37 38 39

FIG. 40. Case 11 (ME 50–74). Male, 6 weeks old. Found dead. Pharynx: Hyperemia, edema, and mononuclear cellular infiltration extending into the muscle. × 104.

FIG. 41. High power view of a portion of Figure 40, through a zone of interstitial myositis. × 205.

40



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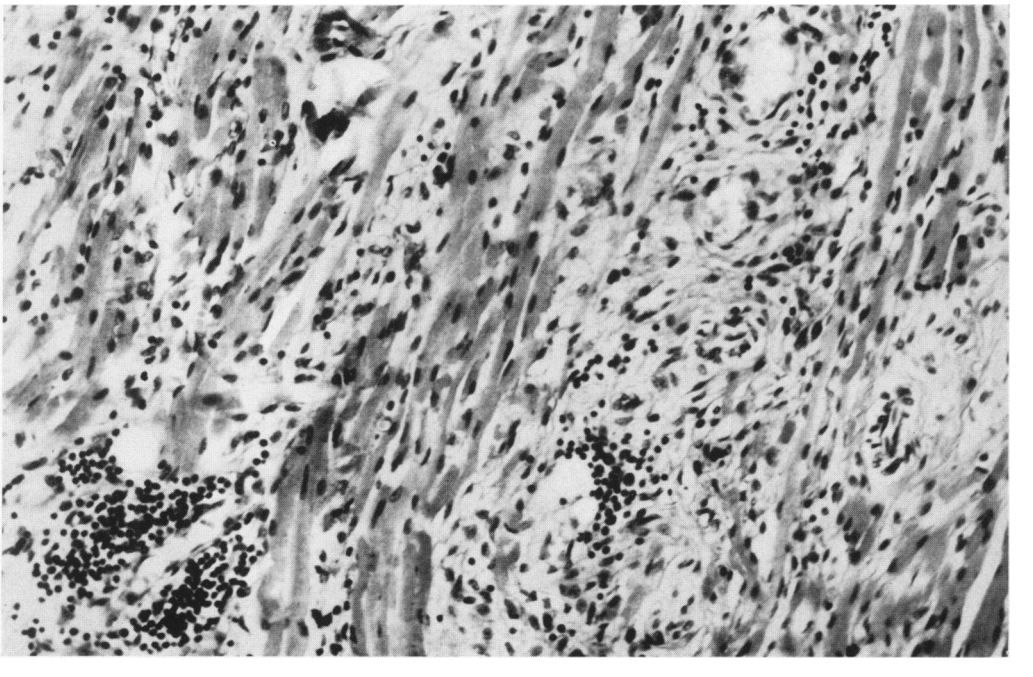
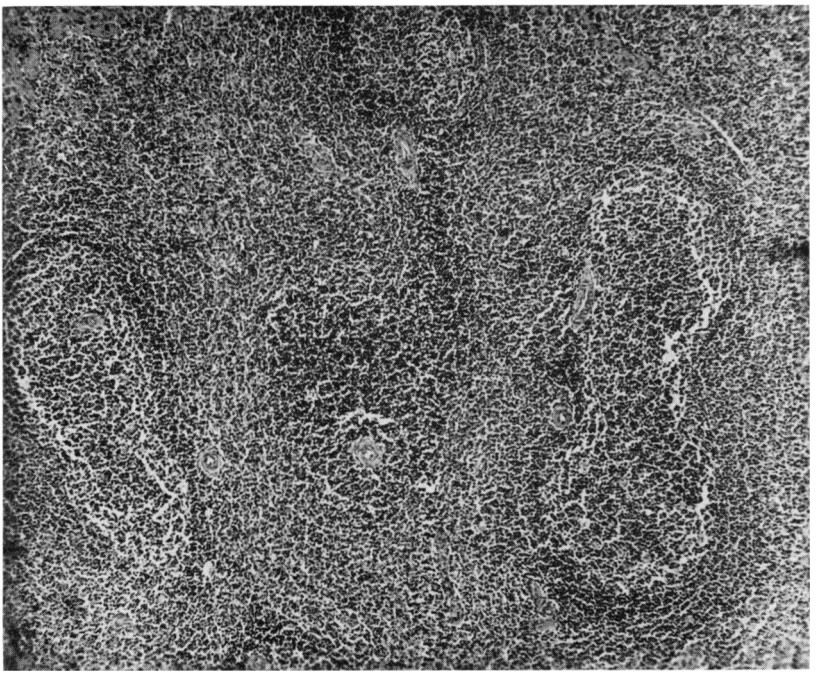


FIG. 42. Case 6 (ME 50–28). Male, 9 months old. Found dead. Spleen: Loss of distinct follicular borders. × 66.

FIG. 43. ME 45–75. Male, 9 months old. Death by drowning. Spleen: Normal follicular borders. For contrast with Figure 42. × 66.

42



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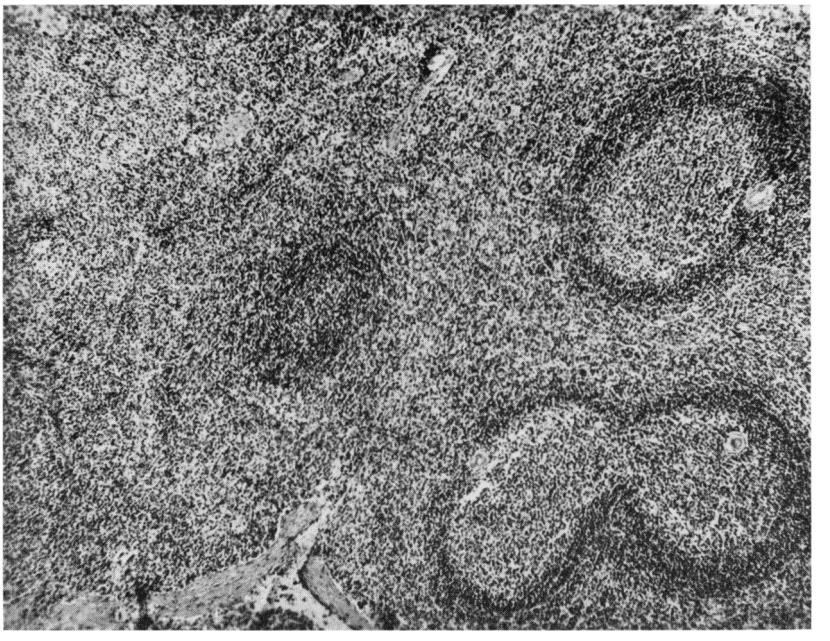
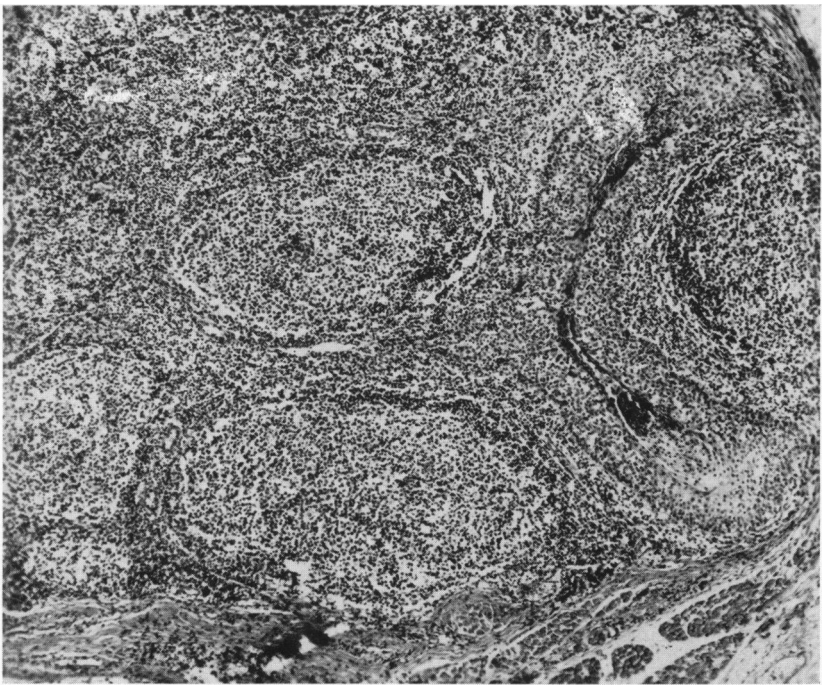


FIG. 44. Case 20 (ME 50–168). Male, 2½ months old. Found dead. Tonsil: Indistinct follicular borders. × 66.

FIG. 45. ME 46–101. Female, 2½ months old. Death by drowning. Tonsil: Distinct follicular borders in normal infant. × 66.

44



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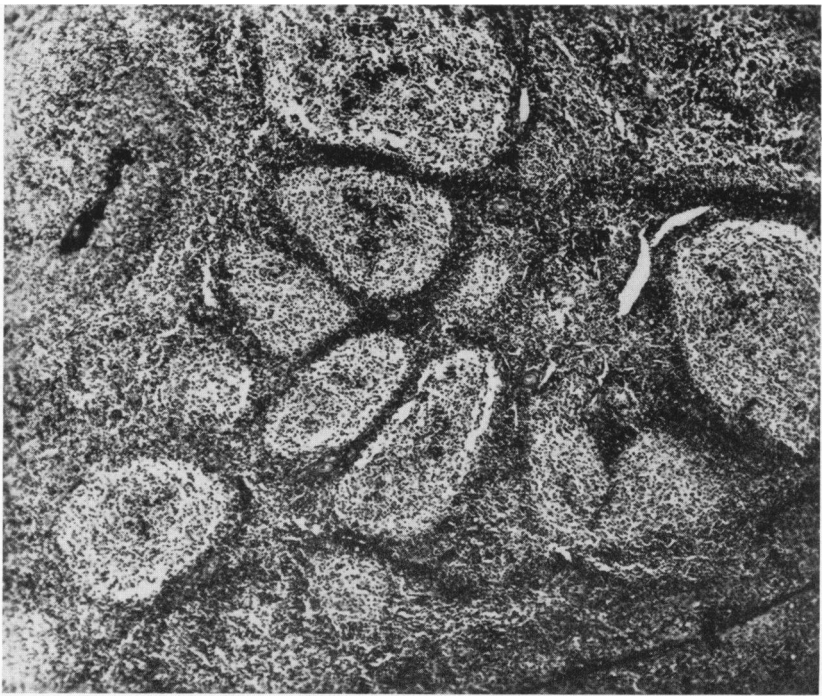
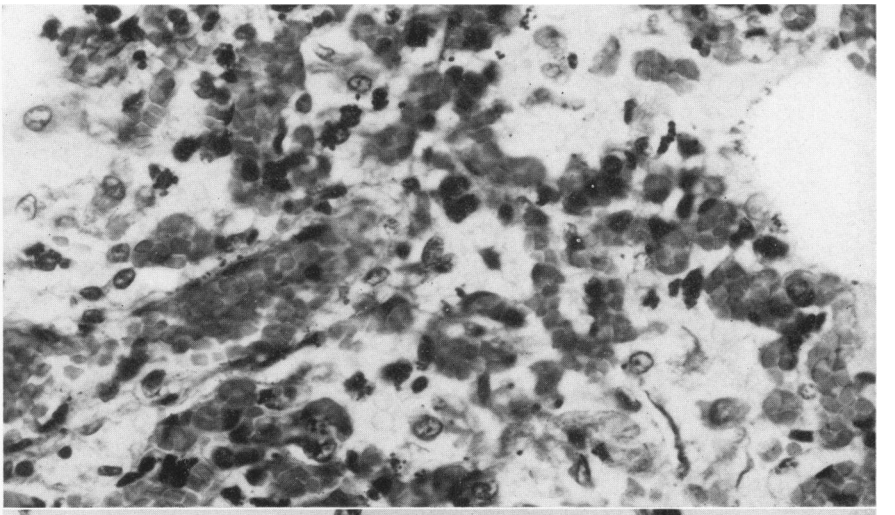


FIG. 46. Case 5 (ME 50–14). Female, 3 weeks old. Found dead. Lung: Extreme capillary congestion. There are a few macrophages in the alveoli. Of note are the squames (from vernix). The dark amorphous material is formalin-produced pigment. × 460.

FIG. 47. ME 46–101. Male, 2½ months old. Death by drowning. Lung: Normal. For comparison with Figures 46 and 48. × 460.

FIG. 48. Case 11 (ME 50–74). Male, 6 weeks old. Found dead. Lung: Extreme congestion and intra-alveolar hemorrhage; macrophages are seen in the alveoli. The dark amorphous material is formalin-produced pigment. × 460.

46



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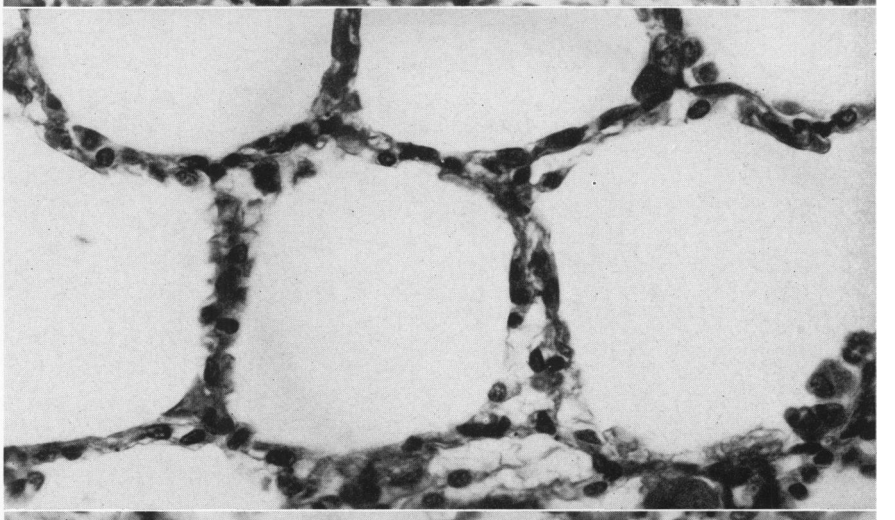

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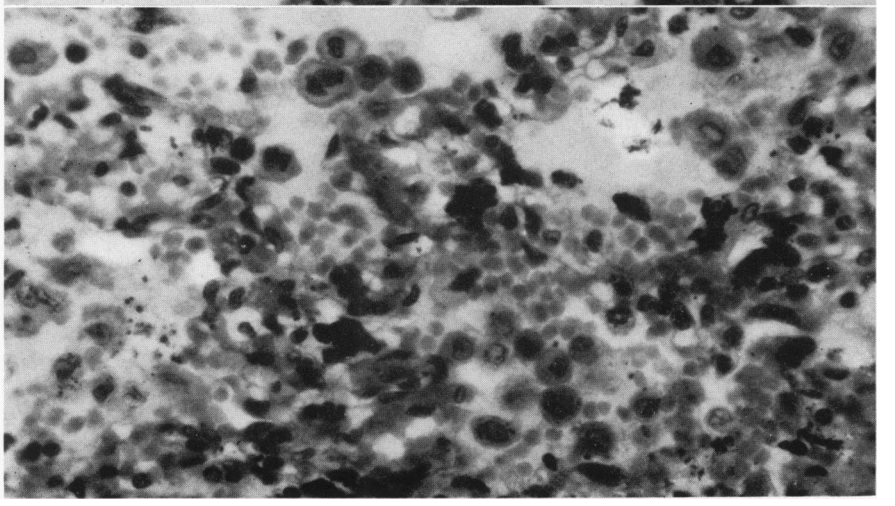
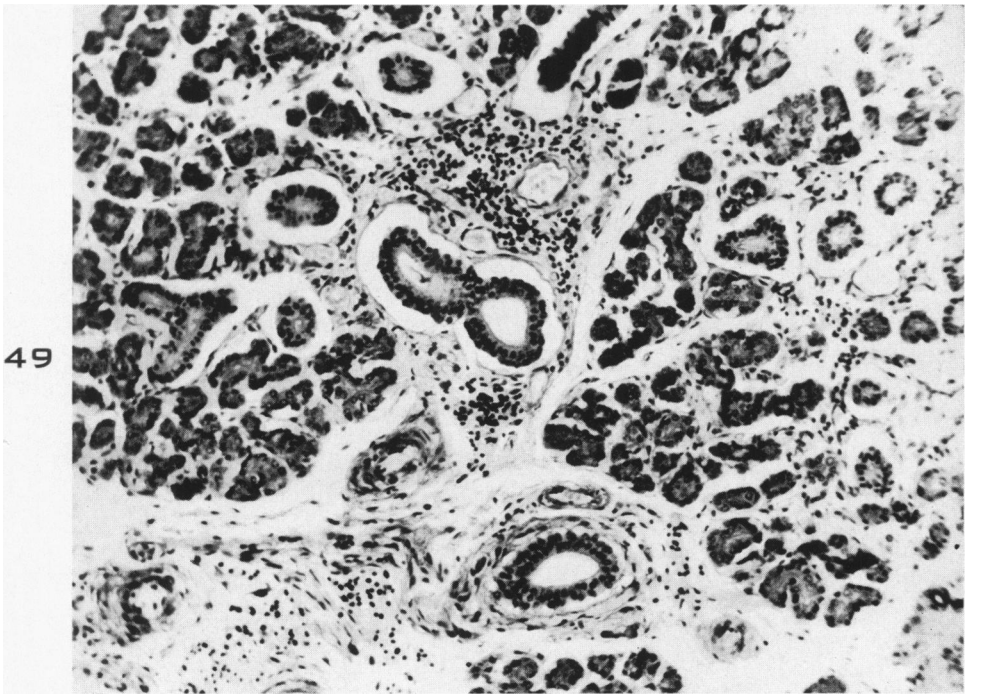


FIG. 49. Case 11 (ME 50–74). Male, 6 weeks old. Found dead. Submaxillary gland: Focal mononuclear cellular infiltration. × 100.

FIG. 50. Case 25 (A 4250). Male, 4 months old. Found dead. Testis: Focal mononuclear cellular infiltration, mainly perivascular. × 205.



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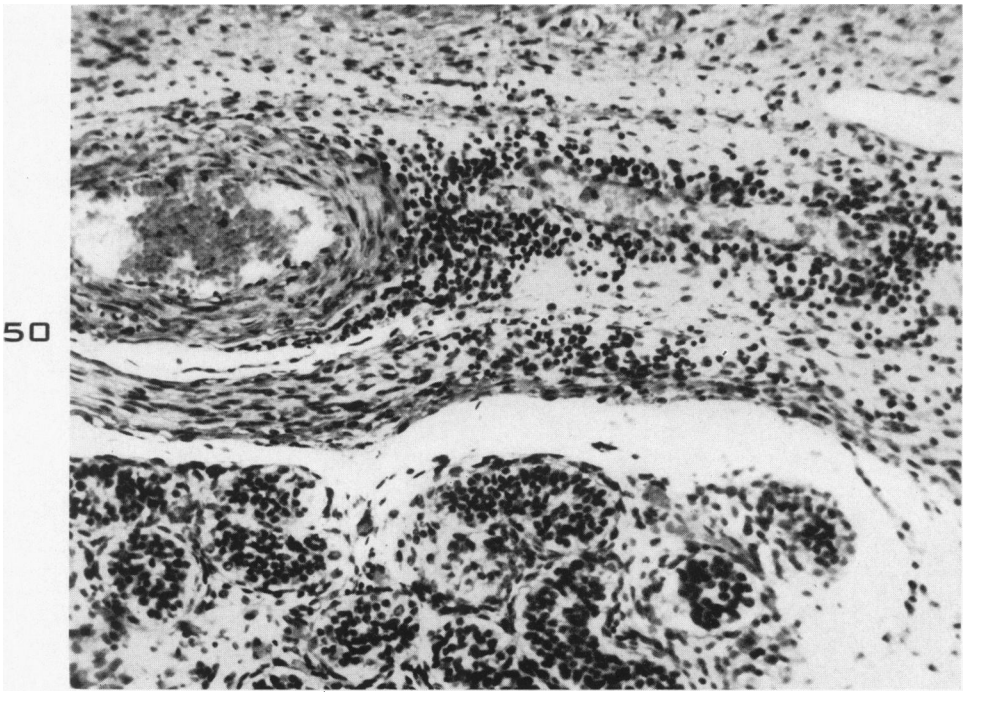

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